Journal of Abnormal Psychology
November 1998 Vol. 107, No. 4, 671-675

Age of Onset for Binge Eating and Purging During Late Adolescence
A 4-Year Survival Analysis

Eric Stice
Department of Psychiatry Stanford University
Joel D. Killen
Department of Psychiatry Stanford University
Chris Hayward
Department of Psychiatry Stanford University
C. Barr Taylor
Department of Psychiatry Stanford University

ABSTRACT

This prospective study examined age of onset for binge eating and purging among girls during late adolescence and tested whether dieting and negative affectivity predicted these outcomes. Of initially asymptomatic adolescents, 5% reported onset of objective binge eating, 4% reported onset of subjective binge eating, and 4% reported onset of purging. Peak risk for onset of binge eating occurred at age 16, whereas peak risk for onset of purging occurred at age 18. Adolescents more often reported onset of a single symptom rather than multiple symptoms, and symptoms were episodic. Dieting and negative affectivity predicted onset of binge eating and purging. Findings suggest that late adolescence is a high-risk period for onset of bulimic behaviors and identify modifiable risk factors for these outcomes.

This study was supported by National Institute of Mental Health Grant MH45431, National Institute for Child Health and Development Grants HD24240 and HD24779, and a postdoctoral fellowship (MH19908) from the National Institute on Mental Health, as well as by the Stanford Center on Adolescence, the W. T. Grant Foundation Faculty Scholars Award, and the McKnight Foundation. Thanks go to Helena Kraemer and Anne Vardy for their thoughtful comments regarding earlier drafts of this article. We also thank Anne Blair-Greiner, Darby Cunning, Diane Strachowski, Suzanne Taborski-Barba, and the Santa Clara Unified and Fremont Union school districts for their help in conducting this study.
Correspondence may be addressed to Eric Stice, Department of Psychology, University of Texas, 330 Mezes Hall, Austin, Texas, 78712.
Electronic mail may be sent to stice@psy.utexas.edu
Received: May 17, 1997
Revised: April 13, 1998
Accepted: April 14, 1998

Little is known about the timing of onset for eating pathology. Past studies using clinical samples have suggested that the modal age of onset for bulimia nervosa is 18—19 years of age (Fairburn & Cooper, 1984b ; Mitchell, Hatsukami, Pyle, & Eckert, 1986). However, because most bulimics never seek treatment (Fairburn, Welch, Doll, Davies, & O'Connor, 1997 ; Whitaker et al., 1990), findings from

clinical samples may not generalize. Moreover, these studies have relied on retrospective reports, which possess questionable reliability. Finally, although studies typically examine clinical levels of bulimia nervosa, little is known about the timing of onset for *specific* bulimic symptoms (binge eating and purging). Scholars have recently called for an examination of the full spectrum of eating disturbances rather than just diagnostic levels of pathology (Smolak, Levine, & Striegel-Moore, 1996). Indeed, half of the presenting cases at eating disorder treatment clinics are diagnosed with partial rather than full syndrome eating disorders (Herzog, Hopkins, & Burns, 1993 ; Williamson, Gleaves, & Savin, 1992). Whereas early clinical studies have suggested that onset of binge eating precedes onset of purging by about 1 year (Fairburn & Cooper, 1984a ; Johnson, Stuckey, Lewis, & Schwartz, 1982), researchers have not examined timing of onset using a prospective design with a community sample. Knowledge about the periods of greatest risk for onset of bulimic symptoms during adolescence may help determine the optimal timing for future etiologic studies.

The dietary restraint and affect-regulation models of bulimia represent the two major etiologic theories for this disorder (Heatherton & Baumeister, 1991 ; Polivy & Herman, 1985 ; Stice, 1994). According to the restraint model, caloric deprivation results in an elevated risk for binge eating. The affect-regulation theory posits that people binge eat to control their negative emotions. The dual-pathway model asserts that the restraint and affect-regulation pathways are the final proximal mechanisms by which psychosocial processes promote bulimia (Stice, 1994). However, few studies have demonstrated that these two factors predict bulimic pathology. Although research has found that dieting and negative affect are positively correlated with future bulimic pathology (e.g., Kendler et al., 1991 ; Leon, Fulkerson, Perry, & Early-Zald, 1995), because these studies did not control for initial symptoms, these findings may simply reflect baseline associations between predictors and criteria. Nonetheless, there is some evidence that dieting and negative affect predict onset of bulimic pathology among asymptomatic individuals (Patton, 1988 ; Stice & Agras, 1998).

Accordingly, we examined the timing of onset for binge eating and purging during late adolescence and tested whether dieting and negative affectivity predicted onset of these behaviors by using survival analysis. This analytic technique models the time to the onset of a discrete event (Willett & Singer, 1993). On the basis of past clinical findings, we predicted that peak risk for onset of binge eating would occur about 1 year before peak risk for onset of purging. We also hypothesized that dieting and negative affectivity would predict onset of bulimic behaviors. Adolescents were followed from about age 14 through age 19 because available data from clinical studies have suggested that the modal age of onset for bulimia is 18 (Mitchell et al., 1986), which implies that this age range represents the period of *greatest risk* for onset of bulimia. We also improved on past research by using a large community sample, structured psychiatric interviews to assess bulimic behaviors, and a prospective design.

Method

Participants and Procedures

At baseline the sample consisted of 543 female students from three California high schools (ninth graders), with the following age distribution in years: 1% were 13—14, 56% were 14—15, 40% were 15—16, and 3% were 16—17 (M = 14.9). [1] Because bulimia is rare among boys (Kendler et al., 1991), we included only girls. The sample was composed of 22% Asians, 3% Blacks, 16% Hispanics, 6% Native Americans, 45% Whites, and 8% who specified mixed racial heritage or "other." Maximum parental education ranged from less than high school (5%) to graduate degrees (28%), which was also the mode.

The study was presented as an investigation of student health behaviors. A passive consent procedure

was used wherein a letter describing the study was sent home to parents, who were asked to return a signed letter or to call the school or investigator if they did *not* want their children to participate. Baseline and three annual follow-up assessments were conducted by trained research assistants during the spring of each year. At each assessment, students completed a self-report questionnaire, had their weight and height measured, and completed a structured clinical interview. Special numbers were used to track participants to ensure confidentiality. In all, 95% of eligible students participated in the study. The average annual attrition rate was 15%. [2] Most attrition was due to students moving (10%), but the remainder was due to absenteeism (3%) and refusal to participate (2%).

Measures Dietary restraint.

Restraint was assessed with the Restraint Scale (Herman & Polivy, 1980) at baseline (sample item: "How often do you diet?"). Because one outcome in the present study was binge eating, two items referring to binge eating were omitted to avoid criterion confounding (α = .74).

Negative affectivity.

Affectivity was measured with eight items from Buss and Plomin's (1984) Emotionality Scale at baseline (sample item: "I get emotionally upset easily"; α = .82).

The Eating Disorders Examination.

The EDE, a structured interview assessing criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM—III—R* ; American Psychiatric Association, 1987), was adapted for use with adolescents. The EDE has acceptable reliability (interrater agreement = .83) and discriminant and concurrent validity (Fairburn & Cooper, 1993). Female interviewers from graduate schools in counseling psychology attended 16 hours of training, wherein criteria for eating disorders were reviewed, a video of a simulated interview was observed, and interviews were role played. Actual interviews were completed in private settings at schools. Students were told that interviews were confidential unless the student disclosed information involving imminent danger to self or others. Interviews were periodically supervised, and feedback was given throughout the study to maintain form accuracy and diagnostic appraisal. The interview consisted of questions assessing binge eating, compensatory behaviors, and weight and shape concerns during the last 3 months. *Objective binge eating* was defined as consumption of a large amount of food over a short period of time, accompanied by a perceived loss of control over eating. *Subjective binge eating* reflected consumption of an amount of food over a short period of time that the participant believed was large even though others would probably not agree and that was also accompanied by a perceived loss of control. Detailed accounts of binges were used to determine whether students had eaten an objectively large amount of food (e.g., a bag of cookies) or a subjectively large amount of food (e.g., 2—3 cookies). Interviewers coded whether the amount eaten was objectively or subjectively large on the basis of the amount of food consumed. Purging reflected use of self-induced vomiting, laxatives, or diuretics to control weight or shape.

Results

Of the 543 girls at baseline, 21 (4%) reported objective binge eating, 12 (2%) reported subjective binge eating, and 27 (5%) reported purging. Overall, 48 girls (9%) reported the presence of at least one of these symptoms at baseline. [3] These adolescents were excluded from analyses to ensure that we predicted onset of symptoms. Participants who were already symptomatic did not differ from those who reported onset of symptoms during the study in terms of baseline age, body mass (wt/ht ^{2}), race,

parental education, dietary restraint, and negative affectivity, or regarding frequency of bulimic behaviors once they were symptomatic (all *p* s > .05).

Of the remaining participants, 24 (5%) reported onset of objective binge eating, 17 (4%) reported onset of subjective binge eating, and 20 (4%) reported onset of purging during the study. In all, 51 adolescents (10%) reported onset of at least one symptom. For those who reported onset of objective binge eating, the frequency over the last *3 months* ranged from "once" to "3—6 times per week," with a mean of "5—6 times." For those who reported onset of subjective binge eating, the frequency over the last 3 months ranged from "once" to "every day," with a mean of "5—6 times." Among participants who reported onset of purging, the frequency over the last 3 months ranged from "once" to "every day," with a mean of "2—3 times." Adolescents usually experienced onset of only one symptom at a time (84%), rather than two (7%) [4] or all three (9%) of these symptoms concurrently. Bulimic symptoms were episodic, in that of the adolescents who reported onset of a symptom and provided data the following year, 94% experienced it only at one time point; just 6% experienced it at more than one time point.

Analyses modeled the time to onset (in years of age) for objective binge eating, subjective binge eating, and purging with Cox's (1970) proportional hazards regression analyses. This analytic technique has the advantage of allowing for varying lengths of follow-up in longitudinal studies and thus minimizes biases due to attrition (Willett & Singer, 1993). Unlike most analytic techniques that require complete data for all time points (e.g., repeated measures analyses of variance), hazard, or survival analysis, uses all available data at each time point because it treats missing data as right censored (i.e., it calculates the hazard for onset only among asymptomatic participants who provided data at each time point). At baseline, participants were free of the symptom used as the criterion in each analysis. The (noncumulative) hazard curve for onset of objective binge eating is shown in the top portion of Figure 1 . This hazard curve depicts the proportion of adolescents who report onset of this symptom, among those who have not yet experienced symptom onset. It indicates that the greatest hazard for onset of objective binge eating occurred at age 16. The cumulative hazard curve for onset of subjective binge eating is plotted in the middle portion of Figure 1 and reveals that the greatest hazard for onset of subjective binge eating was similarly at age 16. Finally, the cumulative hazard curve for onset of purging is shown in the bottom of Figure 1 . Interestingly, the greatest hazard for onset of purging occurred at age 18. [5]

Bivariate Cox's proportional hazards regression analyses indicated that dietary restraint predicted onset of objective binge eating, subjective binge eating, and purging, whereas negative affectivity predicted onset of objective and subjective binge eating (top portion of Table 1). When both risk factors were entered simultaneously, only dietary restraint predicted onset of these outcomes, although there was a trend for negative affectivity to predict onset of subjective binge eating (bottom portion of Table 1).

Discussion

To our knowledge, this is the first study to examine age of onset for specific bulimic behaviors using a community sample and a prospective design during this period. Results suggest that late adolescence represents a high-risk time for onset of bulimic symptoms. Of initially asymptomatic participants, 5% reported onset of objective binge eating, 4% reported onset of subjective binge eating, and 4% reported onset of purging during late adolescence. Altogether, 10% of the participants reported onset of one or more of these symptoms. Collectively, these results suggest that many adolescents experience subdiagnostic levels of bulimic pathology. However, the evidence that these symptoms were episodic indicates that many of these symptoms may be short lived. It was also noteworthy that adolescents more often experienced onset of a single symptom rather than multiple symptoms. These results hint at considerable heterogeneity among adolescents experiencing subdiagnostic levels of bulimic problems.

Results suggest that the period of greatest risk for onset of binge eating was about 2 years before the period of greatest risk for onset of purging during late adolescence. Perhaps it takes approximately this long for adolescents who have initiated binge eating, presumably because of intensive dieting, to abandon dieting in favor of more extreme weight control efforts such as vomiting. Alternatively, it may take until approximately 18 for the normative weight-gain that occurs during adolescence (Hammer, Kraemer, Wilson, Ritter, & Dornbusch, 1991) to be great enough to motivate radical weight reduction efforts. The ages of onset reported here were a few years younger than suggested by early retrospective studies (e.g., Johnson et al., 1982). It should be remembered that these age-of-onset estimates were only for those youths who were asymptomatic at baseline and only for the late adolescent period. However, the fact that we excluded participants who were already symptomatic at baseline suggests that the modal age-of-onset estimates might have been even younger if initially symptomatic participants had been included. Studies that follow youths from preadolescence to young adulthood are needed to provide optimal estimates of the peak risk for onset of bulimic symptoms.

Interestingly, bulimic symptoms appeared to be relatively transient during late adolescence because most adolescents who reported a symptom at one assessment did not report it at the next. The evidence that bulimic symptoms are episodic converges with findings from adult samples (e.g., Drewnowski, Yee, Kurth, & Krahn, 1994). These results may suggest that many of these adolescents were experimenting with these bulimic behaviors but did not progress to more serious levels of pathology (i.e., from partial syndrome to full syndrome). Nonetheless, future research should explore the prognostic significance of these bulimic behaviors to assess whether they foretell eating disorders in adulthood. If most bulimic symptoms naturally remit, efforts aimed at prevention might need to focus exclusively on high-risk adolescents (Killen et al., 1993).

This study was also designed to test whether dietary restraint and negative affectivity predicted onset of bulimic symptoms. Bivariate analyses indicated that dieting predicted onset of objective binge eating, subjective binge eating, and purging. It was noteworthy that dieting predicted onset of purging; although theorists have implicated dieting in the onset of binge eating, few have suggested that it is a risk factor for purging. Bivariate analyses also revealed that negative affectivity predicted onset of objective and subjective binge eating but not purging. This was a unique finding, as this is the first known study to find that temperamental negative affectivity predicts emergence of bulimic behaviors. However, the finding that negative affectivity did not predict onset of purging suggests that the affect-regulation model may be specific to binge eating. These findings generally support both the dietary restraint and affect-regulation theories, as well as the dual-pathway model of bulimia. The prospective design provides assurance that the direction of effects were as hypothesized, and the fact that predictors were assessed by means of paper-and-pencil measures, whereas bulimic symptoms were assessed through structured psychiatric interviews, rules out the possibility that the findings were due to method variance. However, in the multivariate analyses, the relation of negative affectivity to onset of objective binge eating became nonsignificant, and the relation of negative affectivity to subjective binge eating became a trend. These findings may suggest that dieting and negative affectivity are correlated, which causes the effects for negative affectivity to become nonsignificant when the unique effects are examined. This is consistent with the notion that dieting contributes to affective disturbance, as well as with the assertion that these two risk factors arise from some common process (e.g., body dissatisfaction). Alternatively, these results may imply that dieting is a more important risk factor for bulimic pathology than is negative affectivity.

Although this study improved on past research by using a 4-year prospective design, multiple data collection methods, and structured psychiatric interviews, there are limitations. First, the fact that we only examined age of onset for these symptoms among initially asymptomatic individuals during late adolescence limits the generalizability of these findings to early adolescence or young adulthood. This was particularly important because numerous adolescents were already symptomatic at baseline. Future

studies should examine the timing of onset for bulimic symptoms over a broader age range. Second, the relatively small number of adolescents who reported onset of bulimic behaviors may limit the reliability of the age-of-onset and prevalence estimates. Future research should use larger samples, more frequent assessment, and more reliable measures to provide more accurate estimates of these parameters. Third, because we examined only girls, our results may not generalize to boys. Future studies should examine these relations among male adolescents. Finally, the moderate annual attrition, typical of school-based research, may have resulted in biased-low estimates of prevalence rates for onset of bulimic behaviors.

Because this is the first known investigation of the onset of specific bulimic behaviors using prospective data and a community sample, the results have important implications. These findings imply that the optimal time to examine risk factors for the emergence of bulimic symptoms during late adolescence may be from ages 16 to 18. An important next step will be to examine age of onset for bulimic symptoms during early adolescence and young adulthood. Relatedly, future research might examine the periods of greatest risk for emergence of risk factors for bulimic pathology, such as dietary restraint. Future studies should also test whether the predictors of bulimic symptom onset are similar to the predictors of bulimic pathology maintenance over time. A better understanding of the timing of onset and growth of bulimic pathology is necessary for an improved understanding of the etiology of this disruptive pathology.

References

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* ((3rd ed., rev.). Washington, DC: Author)

Buss, A. H. & Plomin, R. (1984). *Temperament: Early developing personality traits.* (Hillsdale, NJ: Erlbaum)

Cooper, Z., Cooper, P. J. & Fairburn, C. G. (1989). The validity of the Eating Disorder Examination and its subscales.(*British Journal of Psychiatry, 154,* 807—812.)

Cox, D. R. (1970). *Analyses of binary data.* (London: Methuen)

Drewnowski, A., Yee, D. K., Kurth, C. L. & Krahn, D. D. (1994). Eating pathology and *DSM—III—R* bulimia nervosa: A continuum of behavior.(*American Journal of Psychiatry, 151,* 1217—1219.)

Fairburn, C. G. & Cooper, P. J. (1984a). Binge-eating, self-induced vomiting and laxative abuse: A community study.(*Psychological Medicine, 14,* 401—410.)

Fairburn, C. G. & Cooper, P. J. (1984b). Clinical features of bulimia nervosa.(*British Journal of Psychiatry, 144,* 238—246.)

Fairburn, C. G. & Cooper, P. J. (1993). The Eating Disorder Examination((12th ed.). In C. G. Fairburn & G. T. Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (pp. 317—331). New York: Guilford.)

Fairburn, C. G., Welch, S. L., Doll, H. A., Davies, B. A. & O'Connor, M. E. (1997). Risk factors for bulimia nervosa: A community-based case-control study.(*Archives of General Psychiatry, 54,* 509—517.)

Hammer, L. D., Kraemer, H. C., Wilson, D. M., Ritter, P. L. & Dornbusch, S. M. (1991). Standardized percentile curves of body-mass index for children and adolescents.(*American Journal of Diseases of Children, 145,* 259—263.)

Heatherton, T. F. & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. (*Psychological Bulletin, 110,* 86—108.)

Herman, C. P. & Polivy, J. (1980). Restrained eating.(In A. Stunkard (Ed.), *Obesity* (pp. 208—225). Philadelphia: W. B. Saunders.)

Herzog, D. B., Hopkins, J. D. & Burns, C. D. (1993). A follow-up study of 33 subdiagnostic eating disordered women.(*International Journal of Eating Disorders, 14,* 261—267.)

Johnson, C., Stuckey, M., Lewis, L. & Schwartz, D. (1982). Bulimia: A descriptive survey of 316 cases.

(*International Journal of Eating Disorders, 2,* 3—16.)
Kendler, K. S., MacLean, C., Neale, M., Kessler, R., Heath, A. & Eaves, L. (1991). The genetic epidemiology of bulimia nervosa.(*American Journal of Psychiatry, 148,* 1627—1637.)
Killen, J. D., Taylor, C. B., Hammer, L., Litt, I., Wilson, D. M., Rich, T., Hayward, C., Simmonds, B., Kraemer, H. & Varady, A. (1993). An attempt to modify unhealthful eating attitudes and weight regulation practices of young adolescent girls.(*International Journal of Eating Disorders, 13,* 369—384.)
Killen, J. D., Taylor, C. B., Hayward, C., Haydel, K. F., Wilson, D. M., Hammer, L., Kraemer, H., Blair-Greiner, A. & Strachowski, D. (1996). Weight concerns influence the development of eating disorders: A 4-year prospective study.(*Journal of Consulting and Clinical Psychology, 64,* 936—940.)
Leon, G., Fulkerson, J., Perry, C. & Early-Zald, M. (1995). Prospective analysis of personality and behavioral vulnerabilities and gender influences in the development of disordered eating.(*Journal of Abnormal Psychology, 104,* 140—149.)
Mitchell, J. E., Hatsukami, D., Pyle, R. L. & Eckert, E. D. (1986). The bulimia syndrome: Course of the illness and associated problems.(*Comprehensive Psychiatry, 27,* 165—179.)
Patton, G. C. (1988). The spectrum of eating disorder in adolescence.(*Journal of Psychosomatic Research, 32,* 579—584.)
Polivy, J. & Herman, C. P. (1985). Dieting and binge eating: A causal analysis.(*American Psychologist, 40,* 193—204.)
Smolak, L., Levine, M. & Striegel-Moore, R. (1996). *The developmental psychopathology of eating disorders.* (Mahwah, NJ: Erlbaum)
Stice, E. (1994). Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanisms of action.(*Clinical Psychology Review, 14,* 633—661.)
Stice, E. & Agras, W. S. (1998). Predicting onset and cessation of bulimic behaviors during adolescence: A longitudinal grouping analyses.(*Behavior Therapy, 29,* 257—276.)
Striegel-Moore, R. H., Schreiber, G. B., Pike, K. M., Wilfley, D. E. & Rodin, J. (1995). Drive for thinness in Black and White preadolescent girls.(*International Journal of Eating Disorders, 18,* 59—69.)
Whitaker, A., Johnson, J., Shaffer, D., Rapoport, J., Kalikow, K., Walsh, B., Davies, M., Braiman, S. & Dolinsky, A. (1990). Uncommon troubles in young people: Prevalence estimates of selected psychiatric disorders in a nonreferred adolescent population.(*Archives of General Psychiatry, 47,* 487—496.)
Willett, J. B. & Singer, J. D. (1993). Investigating onset, cessation, relapse, and recovery: Why and how you can use discrete-time survival analysis to examine event occurrence.(*Journal of Consulting and Clinical Psychology, 61,* 952—965.)
Williamson, D. A., Gleaves, D. H. & Savin, S. S. (1992). Empirical classification of eating disorder not otherwise specified: Support for *DSM—IV* changes.(*Journal of Psychopathology and Behavioral Assessment, 14,* 201—216.)

1

The present study differs from a previous one that used data from the same sample (Killen et al., 1996) in that the former focused on the timing of onset for *specific bulimic symptoms,* and the latter focused on the risk factors for the clinical *syndrome of bulimia.* In addition, this study used only data from the first cohort of ninth-grade students, rather than both ninth-grade cohorts, because only the first cohort was followed for the full 4-year period.

2

Attrition analyses indicated that adolescents who dropped from the study did not differ significantly

from those who completed the study in terms of body mass, race, or bulimic symptomatology at baseline. However, adolescents who dropped were significantly older (*M* = 15.0 years old vs. *M* = 14.8 years old) and reported higher parental education (mode of college graduate vs. mode of graduate degree) than were completers. Nonetheless, this attrition effect only accounted for 5.4% of the variance in age and 5.1% of the variance in parental education.

3

The age distribution (in years) for the 48 participants who were already symptomatic at baseline was as follows: 2% were 13—14, 50% were 14—15, 46% were 15—16, and 2% were 16—17.

4

Of the participants who experienced onset of two symptoms simultaneously, 1 reported onset of both objective binge eating and subjective binge eating and 3 reported onset of both objective binge eating and purging.

5

Because there is some evidence of racial differences in bulimic pathology (e.g., Striegel-Moore, Schreiber, Pike, Wilfley, & Rodin, 1995), we tested whether the hazard curves for onset of objective binge eating, subjective binge eating, and purging were different for Asian, Caucasian, and Hispanic participants (there were insufficient cell sizes for the other racial groups). However, Kaplan—Meier survival analyses indicated that there were no significant differences in the hazard curves for these three racial groups.

Table 1.

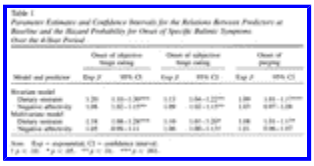
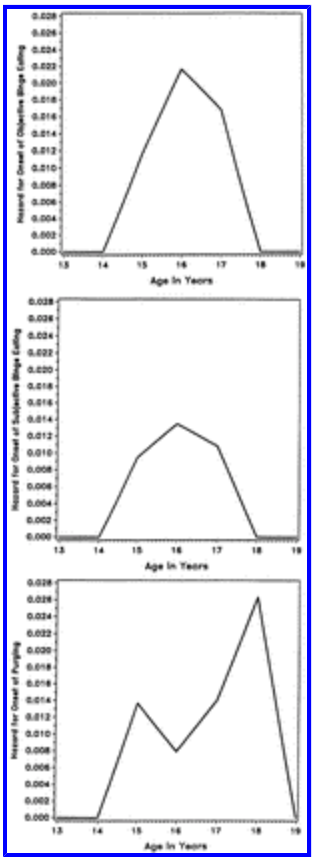


Figure 1. Noncumulative hazard curves for the onset of objective binge eating (top), subjective binge eating (middle), and purging (bottom).


Hazard for Onset of Objective Binge Eating
Age in Years
Hazard for Onset of Subjective Binge Eating
Age in Years
Hazard for Onset of Purging
Age in Years